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OF

THE NEW YORK ACADEMY OF MEDICINE

INCORPORATED 1851

JULY, 1935

PUBLISHED MONTHLY BY

THE NEW YORK ACADEMY OF MEDICINE

2 East 103 Street

New York, N. Y.

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BULLETIN OF
THE NEW YORK
ACADEMY OF MEDICINE

VOL. 11 JULY, 1935 No. 7

Symposium: Angina Pectoris, With Special Reference to Coronary Artery Disease[1]

GENERAL CONSIDERATIONS[2]

EMANUEL LIBMAN
New York

In the time at our disposal this evening, it is manifestly impossible to give anything like a complete exposition of the part of the subject allotted to me. Therefore I will confine myself to clarifying some points of view, and to taking up briefly some of the special aspects that have interested me during the last thirty-five years.

What is "angina pectoris"? When Heberden introduced this designation he did not know the cause of the condition which he was describing. It is advisable, I believe, if the term is to be employed at all, to use it in that way. One should speak only of "angina pectoris due to ?," and when the cause, in a given case, is located, the expression "angina pectoris" should drop out. If I were asked to make a definition, I would say that "angina pectoris" is a term used to describe a pain and certain accompanying clinical phenomena, of a kind that lead one to believe that the causation lies in the cardiovascular apparatus of the thorax, this including the heart, blood vessels, and nerves.

[1] Presented before the Stated Meeting of The New York Academy of Medicine, March 7, 1935.

[2] Dedicated to Professor Anton Ghon on the occasion of his seventieth birthday anniversary.

There are many conditions that may give rise to a pain resembling, or intensifying, an already existing "angina pectoris". These conditions may be of a toxic or a weakening nature, or both. The toxic factors may be bacterial or non-bacterial in origin. Among the latter may be included such influences as tobacco, lead, metabolic disorders. The bacterial causes may be in the nature of a systemic infectious disease, or of a localized infection, such as focal infections and intoxication from the alimentary tract. Of particular interest is influenza, which at times brings about what may be called a neuromyocardial disturbance. There may be present precordial pain and discomfort, and a general feeling of weakness and fatigue, lasting even for as long as three to four years, without any evidence of myocardial insufficiency. Rheumatic fever apparently may act at times in the same way as influenza, but as it is usually accompanied by well-recognized organic changes in the myocardium and blood vessels, it will be discussed later.

Among weakening causes are obesity, anemia, malnutrition, intoxication from the intestinal tract and other organs, etc., etc. While tuberculosis may act as a sensitizing agent, it is by its weakening influence that it more commonly leads to pain in the cardiac area or to intensifying the action of another cause of such pain. Various disturbances of the nervous system may also give rise to pain resembling "angina pectoris", for example, neurocirculatory asthenia, other neuroses, worry, strain, excitement, sexual disorders. The endocrine organs play a great role, especially the thyroid gland and the ovaries. The menopause is one of the most marked sensitizers.

I can refer to but few of the various disorders in the chest, of the skin, muscles, bones, esophagus and mediastinum, that may be productive of precordial pain. It is not generally realized that diseases of the pulmonary artery may give rise to a pain resembling that due to aortic disease. Frugoni, of Rome, who has specially studied this subject, calls the "angina pectoris" due to disease of the aorta and coronary arteries, dolor pallida, and that due to the pul-

monary artery, dolor caerulea. This designation is of value in emphasizing the cyanosis of pulmonary artery disease.

Of particular interest are the pains due to affections of the intercostal nerves, the spine, the subacromial bursa, and other structures about the shoulder girdle. It is not generally known that pain due to a cervical "neuritis" or spondylitis may radiate, not only to the precordium, but as far down as the lower ribs. Pain due to subacromial bursitis often radiates to the precordium (more often, the upper part), spontaneously or on raising or rotating the arm. Such movements of the arm may be of value in making a differential diagnosis. Of similar value, is the method which I have described for the relief of pain due to disorders of the shoulder girdle, especially subacromial bursitis.

Spondylitis, intercostal neuralgia (neuritis ?) and disorders of the shoulder girdle, especially bursitis, may be present together with a real "angina pectoris," and may intensify the latter. I believe that the reason for the frequent coexistence of these conditions is that they essentially have the same origin, namely, a metabolic disturbance with or without secondary focal infections. It is interesting to note how often a subacromial bursitis will start to give pain within a short time after a coronary thrombosis has occurred. At times when pains are present in both arms, the pain in the left side is due to the "angina pectoris" and that on the right, to a subacromial bursitis. An interesting case in connection with what I have just been saying, is that of a man who came under observation suffering from ventricular extrasystoles. He also presented furunculosis of the chin. On the administration of calomel, both promptly disappeared. Somewhat later he complained of pain in the precordium, and pain in the lower ribs, on the left side. On examination, a patch of eczema was found in one palm. Pressure on the cervical spine, at Erb's point, reproduced the pains described by the patient. Following a dose of calomel, and the use of alkalis, all the symptoms disappeared.

The diaphragm, as is well known, may be the source of pain resembling "angina pectoris." The resemblance is often close, because when pain is produced by diaphragmatic disturbances, anxiety, palpitation and cardiac irregularities are apt to be present. Conditions in the diaphragm that may cause pain are hernia (especially the esophageal type), eventration, pressure upwards, tension and inflammation.

In the abdomen, many organs may be the seat of a pain resembling "angina pectoris." Of particular interest, are the stomach, gallbladder, pancreas, intestinal tract, kidneys and ovaries. I will refer only to the colon, especially the descending part and the sigmoid flexure. These frequently give rise to pains that radiate to the precordial area, and even the shoulder and upper arm. Such pains may be accompanied by anxiety, palpitation and arrhythmia. The whole gastrointestinal tract is a great sensitizer of the circulatory apparatus.

Patients suffering from valvular defects (particularly rheumatic aortic insufficiency in the adolescent period), acute or chronic pericarditis, aneurysm, or myocardial disease, may develop precordial pain with less or more radiation, but we do not refer to such pain as "angina pectoris." We simply say the patient has such and such a lesion and has pain due to it. Pain may also occur in connection with attacks of paroxysmal tachycardia even when not referable to coronary artery disease. It is not realized that calcific obstruction of the abdominal aorta may also cause a pain like "angina pectoris." It is important to remember that patients having any of the conditions just mentioned may suffer pain not due to them, but originating in one of the ways now to be described, the valvular defect or other lesion perhaps acting as a sensitizing agent.

The real usual causes of what we call "angina pectoris" are:

1. Hypertension. Here the pain is apt to have wide radiations (up to the side or back of the head), and, at least at

first, to be brought on by exercise. The pains are not apt to be associated with nausea and vomiting.

2. Atherosclerotic dilatation of the arch of the aorta, or more simply, Hodgson's disease. In such patients, there is present a dilatation of the aortic arch, marked left ventricular hypertrophy, and a ringing, at times metallic, aortic second sound. Even when the coronary arteries become narrowed or occluded, and the hypertrophy decreases, a pulsation can still be felt in the jugular notch and the second sound still shows some accentuation.

3. Diseases of the coronary arteries. The important conditions are narrowing or closure of orifices or lumens, and coronary thrombosis.

It is important to remember that patients suffering from narrowing or closure of an orifice, may develop sudden attacks like those due to fresh coronary thrombosis, including nausea and vomiting. Sudden death may occur. It is also to be noted that a fresh infarction may be found in the absence of a recent coronary thrombosis, due to gradual obliteration of a number of smaller trunks.

The lesions usually found in the coronary arteries are atherosclerosis with more or less calcification, or almost pure calcification. The role of syphilis is mainly in causing disease of the aortic wall with occlusion of orifices. Rheumatic fever may cause arteritis of the aorta and/or innominate artery, narrowing or occlusion of orifices, narrowing or closure of the coronary arteries by proliferation, or coronary thrombosis. Other infections cannot now be discussed.

4. The nerves. This is an important subject that has been too much neglected. It is important to keep in mind the pathway, and the state of the psyche.

5. Spasm, alone, or secondary to already existing lesions.

6. Combinations of various causes.

As you well know, there have been put forth a number of theories in explanation of the mechanism at play in attacks of "angina pectoris." At present the anoxemia theory is most prominent. There can be no question that anoxemia is the important factor in cases in which the pain is of coronary origin. But I believe that if one studies the subject according to the classification that I have just presented there is some truth in a number of the explanations that have been offered, but for different cases. I present the following for consideration:

In hypertension, per se, we must admit that strain on the wall of the aorta plays a role (theory of Allbutt). There is clinical evidence that in hypertension the role of the nervous system is important. In fact there are cases of hypertension that much resemble hyperthyroidism, elevation of the basal metabolism being absent, or only slight.

In Hodgson's disease, we encounter a number of factors. There is tension on the wall of the aorta because the blood pressure is always elevated (not as much as in essential hypertension, unless combined with it), there are atherosclerotic deposits in the walls, inflammatory areas and scars.

There is available evidence that such deposits in the wall of a vessel may be painful. When the coronary arteries are narrowed, anoxemia plays the main role.

In rheumatic fever we may have arteritis of the aorta, innominate and carotid arteries. There may be scars in the wall of the aorta. Under all these conditions the pain may well be due to an influence on the nervous pathway. I have published notes on areas of tenderness in rheumatic inflammation (verrucous) of the innominate and of the carotid arteries. In one case, in 1925, it was found that pressure upon one part of the innominate artery produced pain in the precordium radiating to the axilla. A few days later the patient began to experience spontaneous attacks, mainly at night. They persisted for about three weeks. For a number of years, pressure in the same location (which must have been scarred) reproduced the pain. This tenderness

first disappeared in 1933. That pressure upon diseased areas in arteries, even calcific ones, may be painful is amply shown by a study of tenderness and pains due to calcific deposits in the wall of the abdominal aorta. When the coronary arteries are narrowed, or closed (by proliferative inflammation, or thrombosis, or both), any pain is to be explained on the basis of anoxemia.

In syphilitic cases, the mechanism will depend upon whether or not coronary stenosis or closure is present. There can be little doubt that syphilitic disease of the aorta can of itself cause pain.

In the cases of narrowing or closure of the coronary arteries, due to atherosclerosis and calcification, we have every reason to explain attacks of pain on the basis of anoxemia. It must be remembered, however, that these cases may be complicated by other conditions.

After closure by a thrombus, anoxemia is again the explanation. As regards symptoms at the time of an acute thrombotic closure, we must consider as possible additional factors, secondary inflammation of the arterial wall, and dilatation above the point of closure. I have some interesting observations on tenderness of arteries, above acute obstructions, that I cannot now detail.

As regards the nerve kind of "angina pectoris," there is evidence to indicate that the nervous pathway may of itself give rise to pain (like any other nerves), or, in a given case, be one of the causes. One of the arguments that has often been brought forward against the coronary origin of "angina pectoris" has been that the symptoms may be out of all proportion to the extent and degree of the coronary artery disease present. This alone must make us think of the pathway. Earlier writers spoke of neuralgia and neuritis of the cardiac nerves. I have already referred to the influence of atherosclerotic infiltration of arterial walls, and of inflammatory processes and scars. There are definite cases in which an individual died in an attack of "angina pectoris," and no lesions were found. Here we must think of the nerves as well as of spasm.

Of great interest in this connection are the following observations:

1. Cases of what may be called "alternating gout," in which attacks of gout alternate with attacks of "angina pectoris."

2. The case of a man, now 82 years of age, first seen by me with his physician, Dr. Leopold Stieglitz, when he was 77 years old. He had an attack of "angina pectoris" lasting two days, with wide radiation. It came on with an attack of gout. Two years later, in 1932, he had another attack of gout, and again, "angina pectoris." The systolic blood pressure at the time of the second attack was 195. Later there were a couple of minor attacks. Diabetes supervened two years ago. On an anti-gout regimen, there have been no further attacks of gout or of "angina pectoris." Electrocardiographic changes were never evident. The blood pressure now is about 135.

3. An important case (which I have already noted in a publication in 1932) is the following:

About six years ago, I saw a delicate woman, 41 years of age, who was suffering from attacks of "angina pectoris" with wide radiation, these attacks being marked even at rest. She had a systolic pressure of 230 and a diastolic of 130. She was considered to be in a very serious condition, and I was called in to decide whether or not an operation should be performed upon the sympathetic nervous system. She had a murmur which was rather interesting and significant. It was a whistling murmur which was most marked to the left of the sternum low down, not heard as well at the apex, transmitted more to the right than to the left. Such a murmur is not uncommonly found in connection with calcification of the mitral ring. Such calcification to my mind signifies a metabolic disturbance of the type which is called "gout" or "calcium gout". It struck me therefore that the patient might be suffering from a metabolic disturbance.

She was treated medically, not with cardiac remedies but with bromides, alkalis by mouth, and later on, alkaline enemas (carbonate of soda). Within a week she lost the pain and was able to walk without any difficulty. When I saw her nine months afterwards I was informed there had been no pain in the cardiac area during the entire period. The blood pressure was then systolic 250 and diastolic 130.

For some time she suffered from a pain in the right side of the abdomen, due to calcific obstruction of the abdominal aorta (the easily palpable area of infiltration has been steadily increasing). At present she is suffering from attacks of cholecystitis, and has none of the old attacks for which I was consulted.

I believe that I have stated enough to demonstrate the importance of considering the nervous pathway in cases of "angina pectoris."

There is much difference of opinion as regards the occurrence of spasm in the coronary arteries. My own opinion is that there is enough evidence available to make us believe that it does occur. Part of this evidence comes from cases of Raynaud's disease. There is particularly one significant case on record of a patient who first suffered spasm of the arteries of one leg, followed by a visual disturbance (the spasm of the retinal vessels was actually observed), and then suffered a typical attack of "angina pectoris" with a fatal result. No lesions were found in the aorta and coronary arteries.

Feil has reported significant observations on patients suffering attacks of "angina pectoris," with electrocardiographic changes present only during the attacks. These taken together with the studies of Pearcy and his coworkers, make it appear that these findings may well be due to spasm. Pearcy found that ligation of the coronary arteries in dogs causes pain and electrocardiographic changes, which disappear directly after the compression is released. Significant in this connection are other studies made by Pearcy and later investigators, that distension of the hollow abdominal organs may cause cardiac irregularities and

alterations in the electrocardiogram. It is reasonable to infer that these results are brought about by spasm in the coronary arteries.

Another possibility must be considered. According to a hypothesis that I have been developing, any toxic focus may cause hyperemia, edema and even hemorrhage, in the various tissues of the body, and particularly where there has been any previous alteration. A toxic focus may cause spasm, particularly in the bile ducts and the gastrointestinal tract, and probably in arteries. Spasm, in turn, may cause hyperemia and edema in various tissues, but apparently of less intensity than that ascribable to toxic foci. It is necessary to make a careful investigation of the possible role of such changes, particularly in the pathogenesis of "angina pectoris."

I cannot take up the subject of combinations of various causes in individual cases. It is too extensive. Not only is there often more than one cause in the heart and blood vessels, but there frequently come into play the influence of disease of other organs (by way of general effects and more often through reflex action), and the state of the mind. I am tempted, however, to make a brief mention of one case, because it is so instructive. A man suffering from hypertension, complained of two kinds of pain. One, evidently an inverse radiation, was brought on only by walking. It radiated from the back over the left shoulder forward to the precordium. It often also radiated to the side of the head, occasionally the occiput. There were no accompanying clinical phenomena. The second pain had been felt for only a short time, six weeks, before consulting me. This pain, which was situated in the precordium, came on at rest, mainly following meals. It did not radiate and was accompanied by dizziness, which was considered as an equivalent to nausea and vomiting, the patient being hyposensitive to pain. Electrocardiographic changes were demonstrated. This second pain was interpreted as being due to coronary artery disease. Later the patient died with symptoms of acute coronary thrombosis. It is of particular interest that

the pain due to the hypertension, which was the severer pain, was a widely radiating one, and was not accompanied by dizziness.

The aneurysms of the heart that follow so often upon thrombosis of the coronary arteries are now receiving more of the attention that they deserve. Impressed by their frequency, I requested Dr. Paul Klemperer to furnish me with the statistics of the department of pathology of the Mount Sinai Hospital, for presentation at an address delivered at a meeting of the Johns Hopkins Medical Society, in February 1931. Of all the data which Dr. Klemperer kindly furnished me, I want simply to note that aneurysms of the anterior wall of the left ventricle were found in 38 per cent of all the patients in whom a large coronary trunk had been occluded. Clinically, there is present suggestive evidence in two-thirds of these cases of aneurysm. The methods that I have found to be of value need not be detailed, because I have described them elsewhere*.

On a number of occasions, during the last years, I have stated my conviction that atherosclerosis and thrombosis of the coronary arteries, and "angina pectoris" of nerve origin are largely of metabolic origin, and that we must stop considering patients from only the standpoint of the heart and arteries. They should be regarded as affected by a general disorder, of which the cardiac manifestations are but one evidence. This conception has always been considered correct for atherosclerosis in general. It is generally accepted to be in general dependent upon mechanical strain, and a metabolic disturbance.

The exact nature of the metabolic disturbance (or disturbances) is not known. Clinical experience leads me to believe that it is the same which underlies so-called gout—irregular and typical. I usually refer to it as "metabolic disturbance due to ?," or "goutiness." The expression "goutiness" is employed only in a clinical sense and has no reference to any chemical theory, for all studies of recent

* Proceedings of the Inter-State Post-Graduate Medical Association of North America, for 1932, page 409.

years point away from the uric acid theory of gout. It seems likely, as Murchison and others long ago claimed, that a disturbance in liver function is significant in the development of the "gouty conditions." Of importance, perhaps, is a disturbance in lipoid metabolism. On another occasion, I will give the evidence that supports such a view. I might mention an observation of importance, made by Dr. Joseph Milgram, who has kindly permitted me to cite it, in advance of his proposed publication. In a case of subacromial bursitis of the supraspinatus tendon, there was aspirated 4 to 5 c.c. of a greasy, completely ether soluble material. It proved to contain 5 per cent of calcium, and 10 per cent of cholesterol, the remainder being lipoid in nature. As you know, analyses made in cases of subacromial bursitis usually show 95 per cent of calcium, and practically no organic matter. Dr. Milgram's observation is so significant, because it was carried out in an early case.

There are many causes of disturbance of liver function. Some of the important ones are heredity, the absorption of toxic substances from the intestinal tract (due largely to improper methods of eating), and the influence of the mind, especially enforced mental activity and worry. It is my opinion, based upon clinical experience, that the development of focal infections is largely dependent upon hepatic dysfunction. I have already indicated one of the ways in which focal infections may favor the development of "angina pectoris."

One is most impressed, perhaps, by the probable metabolic origin of much of "angina pectoris" of nerve origin, and atherosclerosis and thrombosis of the coronary arteries, by a consideration of the conditions with which they are often associated. They belong largely to the disorders which often occur in "goutiness." Some of these are, eczema, urticaria and angioneurotic edema, arthropathies, bursitis, tendinitis, spondylitis, various forms of neuritis, renal calculus, hemorrhoids, asthma, gallbladder disease, furunculosis, pruritus, thrombophlebitis. Diabetes, which has always been considered to be related to the "gouty" diathesis, is not infrequently noted.

Many years ago, Allbutt made a careful study of the family histories of patients under his care in Leeds. In a paper entitled, "Series and Families of Disease," he listed the disorders which he found associated with the "angina family." They correspond, in the main, to those that I have just stated.

Because of the observations just cited, and other considerations, I decided, some years ago, to apply certain methods that had been found of value in the so-called gouty conditions, to the treatment of cases of "angina pectoris." Some of the conditions in which good results had been obtained by me are, sigmoidal spasm, gallbladder disease, spondylitis, subacromial bursitis, sinusitis, hemorrhoids, and certain cases of eczema, urticaria and pruritus.

The main parts of this method of therapy (which does not interfere with the use of other remedial agents), are the use of calomel (sometimes blue mass), the administration of alkali by mouth and rectum (enemas of carbonate of soda) and the rectal implantation of B. coli. The details of the method, and a discussion of variations of it, I must leave for another occasion. The diet is not limited a great deal, the main considerations being that food should not be rapidly ingested, and that the stomach should not be distended.

I am not sure that a complete explanation can be given of the favorable results that have been obtained, in a goodly number of the patients treated along these lines,—in some, really striking. The following suggestions are offered:

In the first place, liver function is improved, and that removes a generally toxic influence. Diminution in intestinal putrefaction increases myocardial efficiency. This is clear to anyone who has observed that patients not the subject of any cardiovascular disorder, may tire on climbing stairs, and have no difficulty after the administration of a dose of calomel. Besides, the improved condition of the intestinal tract results in a diminution or disappearance of the tendency to spasm that has so bad an influence on the circulatory system.

There is a likelihood that alkalinization and perhaps the whole method of therapy makes nerves less sensitive. Occasionally a very good result goes hand in hand with a reduction in blood pressure. There are some cases of hypertension that are apparently brought about by the absorption of toxic products from the intestines. In one case, in which a very good result was obtained, as regards pain on walking, the use of calomel, alkalis and implantation of B. coli, lowered the blood pressure from 190 to 120. At first, the patient could not walk half a block. Later he was able to walk two miles with ease. He died later suddenly (in a foreign country,—of his old coronary thrombosis?). I might note here that Dr. Louis Katz of Chicago, in his experiments on induced anoxemia, found that it required more exertion to bring on pain, when large doses of alkali were administered.

It is not at all uncommon, in cases of coronary artery disease, to find a low basal metabolism. In such cases, real results may not be obtained until thyroid therapy is instituted. This experience indicates that a further investigation is needed of the mechanism of the results obtained by total thyroidectomy and by the use of other methods, the effect of which is apparently due to reduction of the basal metabolism.

Since 1919, on a number of occasions, I have drawn attention to the probability that thrombosis in the coronary arteries occurs largely because of a general thrombotic tendency. This I have called thrombophilia. By this term is meant a tendency to thrombose, independent of any disease near the vessel, and of any preceding vascular inflammation or traumatism. Occasionally such a tendency seems to be hereditary.

The occurrence of thrombosis in the coronary arteries seems to have a relationship to the metabolic disturbance of which I have spoken, and the exact nature of which we do not know. The reason why some patients with the metabolic disturbance have a tendency to hemorrhage, and others to thrombosis, is not known. It is an important subject for study. The idea that there exists a tendency to thrombose,

and that it may have a relationship to metabolic disorders, is not new. On looking up the literature, I found that as long ago as 1883, von Recklinghausen stated that there exists an individual tendency to thrombosis, independent of any circulatory disturbance, due to such metabolic disturbances as diabetes and gout.

Much of what I have said is meant to serve as the basis of a program for the wider study of the subject of tonight's symposium, with special reference to prophylaxis. I cannot refrain from quoting from Pascal: "One often writes things that one can only prove in that one encourages the reader to think about them himself."

THE TREATMENT OF THE PATIENT WITH ANGINA PECTORIS*

HARLOW BROOKS
New York

The most important basic fact for the physician who is called upon to treat a patient suffering from angina pectoris to realize is that angina pectoris is not a disease, but a symptomatic picture which may be caused by a quite divergent basic pathology. He must also recognize the fact that it is but rarely that he is able to strike directly at the basic pathology concerned in angina pectoris, but that he must in most instances treat the symptoms and signs which are in turn dominantly modified by the personality, emotional status and individuality of the patient, even much more so than in most disease conditions. Physical factors are often much subordinated to emotional ones in this complex and the social obligations and psychic reactions of the individual sufferer are usually of equal if not even greater import than anatomical ones. This is preponderatingly a syndrome in which the treatment of the patient outstrips in its importance the management of basic pathology.

As in most other medical conditions there are, however, exceptions to this general rule. These must each be also fully determined and appreciated by the physician. Where the syndrome is produced by syphilitic disease, which appears in but about 4 per cent of the cases in Eastern North America, this basic pathology must of course be directly attacked. The same is also probably true in at least some cases of gouty origin and perhaps also in other conditions. This is, however, the rare exception and not the rule in angina pectoris.

Hence it is that treatment if successful must be also twofold, one type directed to correct or to modify a basic causa-

* Read before the Stated Meeting of The New York Academy of Medicine, March 7, 1935, as part of a symposium: Angina Pectoris, with Special Reference to Coronary Artery Disease.

tive pathology which varies much in individual cases and secondarily to prevent or neutralize inimical emotional factors which strikingly vary also in each individual. We must in every instance comprehend and treat the highly individualistic physical, and especially the emotional characters of each patient.

This syndrome does not permit of a standardized scientifically based treatment, for the individual patient is not standardized but is a very pleomorphic biological and emotional integer.

The subject with angina pectoris is notoriously one sensitized particularly to many emotional factors, often of extremely varied character. Every clinician well realizes that emotional factors and stresses are quite as frequent exciting factors of the syndrome as are the varied physical and chemical conditions which also underlie it.

This is particularly strikingly manifested in those factors which incite or precipitate the attack. An association of emotional and physical exciting factors is almost certain to precipitate attacks in susceptible individuals. We may also say that given the basic mechanism of the condition, emotional states quite as certainly set the process in gear as can physical factors.

Thus it is that treatment, preventive, palliative or curative, must make full acknowledgement of these facts. One individual must differ in these respects as widely as individuals may differ one from another in any other plane, and so also it is certain that each person may differ at different times and under different emotional and physical states as much as in any other of the varied grades of life.

As a rule, and with most persons, emotional states may be more readily controlled and directed than physical ones. We must recognize also that emotional factors often very largely control the physical pictures. Few other organs in the body more strikingly illustrate this fact than does the heart. Anger, fear, love, surprise, even intense joy more strikingly and profoundly influence the heart rate and rhythm than most pathological or physical factors of which

we know. The same is also true of arterial, venous and capillary tension which vary more quickly under such stimuli or sedation than from any anatomically pathological pictures; as to the influence on capillary flow in particular, note the striking blush of embarrassment, the sudden and intense pallor of anger, the appearance of sweat, heat or cold of the skin, nausea and diarrhœa, and the enuresis of emotional origin and the like.

It is a fact that the danger of an attack of angina is in very large part coincident with the activity of the symptomatic manifestation. With the subsidence of the pain and anxiety of the attack, danger rapidly wanes. It is therefore most important that we study and attempt to evaluate these factors which are so necessary in the mechanism to prevent or lessen the angina. To a very large extent, prevention and relief are accomplished by similar type factors or measures, but in direct opposition. Of all these by far the most important is REST. The most complete form of rest, is, of course, sleep. Except in certain cases in which sleep appears to precipitate attacks, probably in accordance with the theory of Allbutt, sleep is one of our most efficient preventives of the onset of attacks. Sleep may be produced in adequacy or even in super-abundance by placid mental states, by the use of such sedatives as the bromides, chloral, the barbiturates, alkaloids of opium, and by many other drugs, among which we must not forget alcohol.

Monotonous reiteration, the reading of poetry, music and the like are all sedatives of real value in selected cases of angina pectoris. Music, usually one of our best sedatives, may, however, in certain individuals so stir the emotions, through keen appreciation, as to precipitate attacks. Mild exercises, intelligent physio-therapy are highly productive of rest in many instances. Electricity may be also employed and in by no means a small number of instances such measures may be found far more effective than drug sedatives.

Diversions, games, congenial conversation, even when some physical effort is entailed often act most effectively as

sedatives. Elimination of worry, however, if it may be accomplished, is often of tremendous value. I have accomplished the elimination of attacks in a good many instances by allowing the patient to return to congenial work. Control of environment, hospitalization when domestic conditions are trying and vice versa, sending patients from the hospital to enjoyable home surroundings has broken the attacks also in a good many instances. It must be always recalled that in the application of the rest element in treatment of cases of angina, the individualism of the patient must be especially considered.

From the mere physical standpoint, rest is most completely accomplished in bed. Many persons, however, particularly those of a restless and introspective character, do very badly on bed rest except when it is given for but limited periods of time and for definite purposes as in exhaustion and the like. Few cases are permanently improved by prolonged bed rest, except where coronary thrombosis has taken place when the physical rest is essential for anatomical reasons, as, so that the scar of the infarcted areas may be healed.

Control of environment is possible in many circumstances where the physician is the family doctor and when he fully retains a proper relationship of entire confidence of his patients, now, unfortunately, a somewhat rare occasion.

Under environment it is the function of the physician to arrange for play and diversion as much as it is to assist the patient to adjust his business and social obligations to affairs which he may carry on, not only without detriment but often with real benefit.

Climatic adjustment is in very many instances a matter of very great importance. Angina pectoris is relatively rare in the tropics and it is less in the subtropics than in the cold and temperate zones. Where it is possible for the patient to spend perhaps only the inclement months in a tropical or subtropical climate, it may be wisely advised. Some patients do better in moist warm climates, such as in Florida and southwestern California, Spain, in India and

the like. Others do best in dry warm climates, as in southeastern California, Arizona and New Mexico, in Egypt, or in parts of Old Mexico.

Altitude may also be advantageously employed not only as regards temperature, but also as regards winds and air pressure. Thus it is that the therapeutist must familiarize himself with many geographical factors, with conditions at sea and the like; all are matters of great import to the patient with angina pectoris. Exercise requirements must be also studied and in considerable part this considers also altitude, days of sunshine and heat. As a general rule patients do not thrive at the higher altitudes and many are at their very best at sea level or on the coast.

The problem of vasodilatation has a good deal of bearing on these matters of climate and altitude. As a general thing patients do not do well at the higher altitudes, and though some are best at medium altitudes the matter as a rule must be worked out experimentally. Allergy, and sensitivity to prevailing foods and to plant life becomes of importance. This especially applies to those numerous persons of marked allergic sensitivity, for these agents also tend toward increasing attacks or bring immunity to them.

As a general rule it will be found that conditions favoring vasodilatation are desirable. Warmth, comfort and congeniality of surroundings are matters of very great import in this respect. Properly adjusted hydrotherapy and physiotherapy of other types are all matters of importance in these respects and much of the benefits achieved through the agency of the sedatives may act through these channels. The value of such drugs as the bromides, chloral, the barbiturates, of paraldehyde and finally of the opium group act in this way. The judicious and timely employment of this class of drugs can be most fittingly applied through many of these agents. Alcohol is one of our standby substances for this purpose and many patients find that they can much mitigate and diminish their attacks by the use of alcohol, especially when administered at bed time or given with the meals. This fact has been fully recognized by many modern

writers on the subject, but it is particularly forcefully emphasized by the observation of Heberden, who first described the condition and who rated alcohol as second only to the opium group in point of its efficiency in this respect.

Lauder Brunton by introducing the use of the nitrites for the relief of acute attacks of the syndrome conferred an ineffable boon on most of the sufferers from angina pectoris. In the average case there is no other drug so prompt and efficient as the nitrites in the abortion of an acute attack unless it be accompanied by a coronary thrombosis in which instance the nitrites are usually of little avail. The nitrites given as a routine in the prevention of attacks is also tremendously beneficial in a very high percentage of cases, as every physician knows.

The control and elimination of the sense of pain is one of the major purposes of treatment in angina pectoris. It has already in large part been suggested in what has gone before but it is exceedingly important for the physician to realize that in most cases with the elimination of pain the danger of the attack is also much done away with. Suggestion and autosuggestion are often of tremendous value in the control of pain in many medical conditions but they are well nigh useless in true angina pectoris.

The control of pain is considerably enhanced by the maintenance of a chronic state or condition of sedation such as we may bring about through the action of the vasodilators, but relief of the suffering of the immediately acute attack can, as a rule, be accomplished except when thrombosis of a coronary has taken place when powerful sedatives and analgesics are demanded, morphine, opium or some of their combinations. Alcohol in massive doses is recommended by Heberden, but we now have other much more quickly acting and more efficient drugs for this purpose.

In most cases the combination of the use of the opiates in acute and severe attacks on a background of other soporifics and sedatives as chloral, paraldehyde or the barbiturates are often well attended. Mere vasodilators such as the

salts of theosine and caffeine as a rule are not very effective for the relief of pain except as preventatives and unless these drugs are combined with routine exhibition of pure sedatives they still have the unfortunate effect of causing considerable nervous and mental stimulation which, in turn, acts to excite the numerous factors which play so large a role in the precipitation of attacks.

The value of these drugs in actual coronary thrombosis or coronary narrowing is, however, very important, as most of us know. It is a curious fact however that many prescribe theosine, caffeine sodium benzoate, metaphylin, theominal, and at the same time cry out against the use of tea and coffee, which are so much more agreeable ways of introducing these very useful drugs.

Some physicians lay great stress on the importance of the dietetic treatment of angina pectoris. As a rule this is very ineffective and not a well based theory except in those instances in which an allergic factor may be introduced through food. In the main it is the quantity of food taken, and the speed of its ingestion which are the more important factors. It is usually the over distention of the stomach and the pressure exerted by the over distended stomach on the diaphragm which precipitates the attack, and not the character of the foods as such. To a certain limited degree, however, this statement may be qualified where there is a salt retention of importance. The salt rich foods, therefore, may require limitation of ingestion. In gouty cases the nitrogen rich foods must be eliminated. In allergic cases, as so many are to tobacco for example, this drug or condiment must be much more limited or entirely eliminated.

For the same reason foods which excite excessive flatulence and colonic distention, as cabbage, turnips, beans, the fats and the like may be wisely much curtailed. Constipation and all manner of abnormal fermentative processes within the gastro-intestinal tract are also conditions which may become of importance, but their role is usually much exaggerated by the lay public and the lay medical advisor.

Though the syndrome depends on actual cardiac pathology without probable exception, acting in most cases in conjunction with a sensitized nervous and sympathetic arc, there are other pathological conditions which may play a role in the occurrence of angina pectoris. Thus emphysema, adhesive pleurisy, pulmonary fibrosis, chronic bronchitis and bronchiectasis, pulmonary and bronchial neoplasms, and the like may become important determining factors.

The same is also true of some endocrine defects as in thyroid disease, either of the hypo- or the hyper-active types. Adrenal disease also probably plays some part in many cases and probably also the sex glands and pituitary disturbances as well. The very interesting and suggestive work which Crile has done in adrenal isolation has impressed the writer greatly.

It is, I am sure, apparent that the treatment of angina pectoris may be a very complex problem indeed, and it is further apparent that intimate individual study is the essential of success in the management of any case.

All too little attention has been paid in the past to the preventive treatment of angina pectoris. Every clinician of experience knows that coronary artery disease, aortitis and most of the conditions which lead on to the production of the syndrome are highly hereditary ones. The syndrome is a definitely familial one, and yet how very little attention has been paid to its prophylaxis.

The records of most active practitioners show all too clearly that heredity is a very important factor in the occurrence of angina and in most families even the specific type of the syndrome is to a considerable extent preserved. How very wise it would be to advise against marriage among members of anginal or coronary disease families, and where children are born to such unions or in such families to teach them from their early years the building up of self control of the emotional tendencies and the like. Prophylactic training against the development of early arteriosclerosis might be easily cultivated, and the early training in ath-

letics, in the habits of study and in the selection of occupation, diversions and the like might readily build up a resistance against the disease rather than to develop a predisposition by environment, diet, habits and customs of life which are well known to favor the evolution of the pathology back of the syndrome.

The time allotted to me this evening for the discussion of the treatment of the patient with angina pectoris is very brief for so important a topic but I should much fail my purpose did I not give some time to the presentation of the operative methods of treatment.

Very briefly stated operations in this condition are done for two quite different purposes. The first to be considered is those procedures which are primarily designed to relieve the suffering and the second is those which are performed with the hope of relief of basic pathology. Both groups of operations are of comparatively recent origin and consequently few clinicians have thus far seen enough conclusive results on which to base a sound prognostic opinion.

Probably the most simple of the procedures designed to relieve the distress of the syndrome, for we all meet with cases in which medicines alone fail us in this respect, is through injection of the nerve roots, substantially by means of nerve block. Probably the most satisfactory method is that of Swetlow. It succeeds in its purpose in the hands of skilful operators, in a very considerable number of cases. It is rarely however permanent in its final results and the danger of death is not averted by this method.

These same criticisms apply in the main also to the operations which involve section of the sympathetic trunks of the neck or the removal of the cervical sympathetic ganglia. There are several operations of this type under study, perhaps the most successful are based on the primary work of Jonnesco. In some cases these methods give great relief of the agony of the syndrome, they do not relieve pathology however, many cases recur and even immediate relief is problematic. Mackenzie among others promptly pointed out that to some extent the danger of death was increased

by these operations, in as much as the warning of pain was blocked and the patient was therefore tempted to undertake more effort because of this fact. My own observation however does not entirely bear out this statement since it appears that in at least some instances a diminution in the nervous reflex exciting attacks is also gained by the operation. Thus far the results have not justified the operation except in cases where the agony was so great that almost any risk was worth the taking. Clinicians in general appear to feel that these operations should be reserved for severe cases only, instances in which very inadequate relief only could be won by rest and by medical treatment.

Of the second group of surgical attack of the disease in which an attempt to remove the causative pathology is made, undoubtedly the most popular method at present is total or subtotal thyroidectomy. That thyroid pathology may play a weighty role in many cases, I think is beyond doubt but that it is universal remains to be proven by results and not theory. Subtotal thyroidectomy has greatly relieved some cases in my observation, in a few it has produced an apparent cure, at least for the time being.

I have not seen enough cases in which total thyroidectomy was performed to give a personal opinion, what I have seen has given me no encouragement, nor has this operation equaled the result achieved by the subtotal method, in my opinion.

Still a newer operation attempts to correct the condition by the establishment of an adventitious circulation. One wonders if it will be more successful than that accomplished in adhesive pericarditis.

For a very few remaining moments I now wish to take up in a somewhat more specific manner the treatment of the individual in the acute attack of angina pectoris. The first object should be to produce a vasodilatation as promptly as possible. This is most readily accomplished by the very prompt administration of nitroglycerine or of amyl nitrite. A little experience with each individual case will soon show

which of these two is the more individually effective. Nitroglycerine is best given in solution, but fresh tested tablets, preferably hypodermic ones are thoroughly satisfactory. They can be crushed by the teeth and swallowed, it is far more satisfactory than the attempt to hold the tablet under the tongue. When amyl nitrite fumes are inhaled the capsule should be crushed in the handkerchief and a concentrated gas inhaled if the best result is expected. Neither of these drugs is dangerous in itself, though to withhold them is often highly so.

If prompt relief of pain is experienced, no repetition is indicated, but if otherwise it may be repeated or a general sedative taken, such as chloral, a barbiturate, amytal or the like. The second object is to give a sedative which is also a slower acting vasodilator. Morphine is best but for obvious reasons it is not usually wisely so used. A good drink of a strong alcoholic is often very efficacious. Rest, physical and emotional, and if possible a short period of sleep is very desirable. A very definite effort to self control should be made a part of this treatment, for fright, terror or anger prolong and tend to reexcite the attack. Do not take food, exercise, or engage in any emotional effort until after a short or longer period of rest has been taken.

AN EVALUATION OF THE SURGICAL TREATMENT OF ANGINA PECTORIS*

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A critical appraisal of the different forms of surgical treatment of angina pectoris should properly include a detailed consideration of the theoretical aspects of each type of operation, as well as of the actual results obtained. The time at our disposal is so relatively limited that any discussion of the anatomical and physiological basis must be brief and quite inadequate. Those of you who wish a more comprehensive knowledge of this aspect of the problem may profitably read the excellent series of papers by those authors to whom I shall refer in a moment.

There are three types of operation that have been proposed and tried for the relief of anginal pain: (1) section or removal of a small or large part of the cervical sympathetic nerves or ganglia on one or both sides; (2) section of, or injection of alcohol into, the upper thoracic sympathetic ganglia or the corresponding dorsal nerve roots; and (3) total removal of the thyroid gland. I wish to discuss these briefly in that order.

In 1916 Jonnesco[1] of Bucharest first adopted a suggestion made by Francois-Franck seventeen years earlier. Franck was convinced on the basis of certain experimental observations and his review of cervical sympathectomy performed for various conditions, that these nerves served a sensory function and conveyed pain impulses from the heart to the central nervous system. Jonnesco performed widespread excision of the sympathetic nerves in the neck of a patient who had syphilitic aortitis and angina pectoris, and secured complete relief of pain. Despite this, the operation did not become popular, and in 1923 Coffey and Brown[2] of California suggested a much simpler procedure for the same pur-

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pose, namely, removal of a smaller portion of the sympathetic nerves or ganglia on one side only. Within the next five or six years a great many patients were subjected to this procedure or some modification of it, and when it became clear that unilateral resection was often ineffective, some surgeons returned to the more radical and extensive operation of Jonnesco which became known as cervicothoracic sympathectomy, and consisted of the removal of all three cervical ganglia and the first dorsal or stellate ganglion on both sides. As will appear later, this bilateral operation was based upon beliefs now thought to be erroneous.

In 1933, White[3] summarized the reported cases subjected to one or the other type of sympathectomy, and reported the results as follows: Of one hundred and eighteen cases subjected to upper cervical sympathectomies (Coffey and Brown), 48 per cent had a satisfactory result. Of sixty-six cases subjected to the complete cervico-thoracic sympathectomy (Jonnesco), 53 per cent had a satisfactory result. The operative mortality was from 7.5 to 12.5 for the simpler operation, and about 20 per cent for the complete Jonnesco procedure.

I am sure I need not remind you of the difficulty inherent in this method of appraising results. The degree of enthusiasm of the author may lead him to classify as highly satisfactory a result which impartial observers would regard as far less so, and it is a not uncommon experience, as Dr. Levy[4] has pointed out, to find the tables and conclusions of certain authors at wide variance with the case reports upon which they are based. In other words, even if one accepts the author's own report of his cases, it becomes impossible in many instances to agree with his classification of results. Acknowledging this difficulty, it is interesting to find one of the more enthusiastic of the earlier advocates of sympathectomy speaking thus in 1933:[5] "It seems to us that the results in all of the procedures enumerated and so frequently practised fail to give a sufficiently satisfactory percentage of good results to let us feel that we are on the right track in treating this disorder. . . . It is true also that

the risk of the major operation is something to be considered. We have convinced ourselves that the figures for the various procedures on the sympathetic apparatus in the treatment of angina pectoris are not sufficiently hopeful to justify continued minor perfections in the technique already at hand. Certainly the changes brought about are only of a quantitative measure, and complete relief has rarely been given. It would appear that the basic considerations must be somewhere faulty."

I believe it is correct to state that cervical sympathectomy has fallen into almost complete disuse because increasing experience indicated that the results did not justify the procedure. I have written to a number of those who were at one time interested in this form of treatment, and practically all who have replied indicate that they are no longer advocating it. Since there is now excellent reason for thinking that the operation was based upon incorrect beliefs, it seems to me unnecessary to consider it further.

In 1925, Mandl[6] in Europe, and in 1926 Dr. George Swetlow[7] of this city, reported groups of anginal patients who had been remarkably relieved of pain by the paravertebral injection of alcohol into the dorsal nerve roots on the left side. In these earlier reports the procedure was not standardized as to the number of nerves to be injected; in Swetlow's cases the decision depended in some measure upon the areas of hyperesthesia in the skin that could be discovered by careful protopathic and epicritic sensory tests; from the studies of Head and of Mackenzie, it was thought that these areas would indicate the dorsal root ganglia that were being irritated by the passage of pain impulses from the heart. In the eight patients first reported by Swetlow, alcohol was injected into from two to nine dorsal nerve roots; prompt and satisfactory relief from pain was secured in every instance. In a paper five years later, he reported a group of twenty-two patients, with relief in eighteen of them.

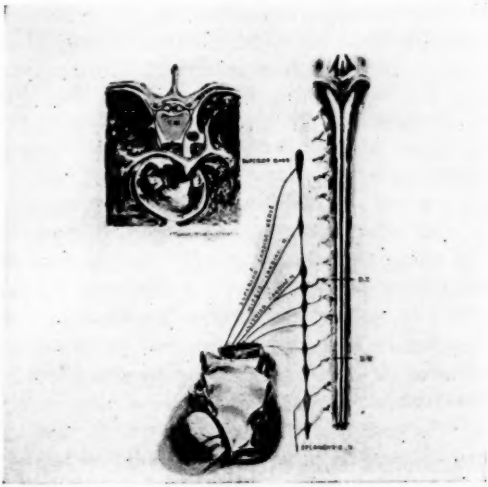
Between 1926 and 1930, observations of fundamental importance in this connection were reported by a number of

different observers [8, 9, 10, 11], who demonstrated the existence of postganglionic fibers leaving the upper two to four thoracic ganglia and running into the posterior cardiac plexus. These were demonstrated in laboratory animals and in man, by anatomical and physiological methods. If it could be shown that these fibers carried sensory or motor impulses which played an important part in the production of anginal pain, it would immediately become obvious why cervical sympathectomy had been unsuccessful, for no operation performed through the neck could sever these structures, and their persistence would account for the continuance of anginal pain after even complete cervical sympathectomy.

With this possibility in mind, James White of Boston and his collaborators[3] performed a series of experiments upon dogs. As an indicator of the occurrence of pain, they employed the method of Sutton and Lueth,[12] passing a ligature about the left anterior coronary artery and leading it out through the wound, which was then closed. Brief traction upon this ligature would cause occlusion of the artery, and the resulting ischemia of the heart muscle invariably caused characteristic changes in the behavior of the animal, stiffening of the limbs, a marked increase in the rate and depth of respiration, and considerable restlessness. In twenty-one animals this was used as an index of the effectiveness of various neurosurgical operations for interrupting the pathways of cardiac pain, or of impulses thought to correspond with pain impulses in man. Their results may be summarized briefly as follows: Cardiac pain was not transmitted by the vagus nerves; bilateral section of these nerves did not alter the characteristic response of the animal to coronary arterial occlusion. If afferent impulses from the areas to which cardiac pain is usually referred were blocked by section of the upper intercostal nerves on both sides, pain was not prevented. Bilateral resection of the stellate ganglia might diminish, but did not block, all sensory stimuli from the heart; a distinct sensory response was obtained in five of six animals in which this test was made.

On the other hand, bilateral removal of the sympathetic ganglia from the stellate down through the fourth thoracic ganglion, or section of the upper five posterior dorsal nerve roots, appeared to interrupt all sensation from the heart. They showed that bilateral excision of the stellate ganglia did not completely destroy the motor pathways to the heart. The upper thoracic ganglia (below the stellate) send important motor as well as sensory fibers directly across the posterior mediastinum to the posterior cardiac plexus. If these are stimulated electrically, the heart rate may increase as much as 58 per cent.

These statements concerning the anatomical and physiological relationships of the heart and the sympathetic system will perhaps be clarified by reference to the illustration. This photograph is of a drawing in a paper by Dr.



James White which appeared in August, 1933.[13] The heart and spinal cord are viewed from the back, and the position of the heart has been shifted to the left for purposes of illustration. The heavy black line represents the sympathetic trunk, beginning with the superior cervical ganglion and showing the successive ganglia down to the

fifth dorsal. The lines connecting these ganglia with the posterior cardiac plexus represent in order the superior, middle and inferior cardiac nerves running from the corresponding sympathetic ganglia, and below these the fibers mentioned as having been described between 1926 and 1930 by a number of different observers. These run directly across the posterior mediastinum and apparently carry both sensory and motor impulses. The middle and inferior cardiac nerves carry both afferent and efferent stimuli, but the superior cardiac nerve is thought to be purely motor. It is important to recognize that all of the cardiac fibers concerned in the conduction of pain impulses converge in the upper thoracic ganglia and then enter the spinal cord over the white communicant rami and the posterior roots. According to our present conceptions, there are no white communicant rami joining the upper cervical ganglia with the spinal cord, so that impulses travelling from the heart over the middle and inferior cardiac nerves must travel downward in the sympathetic chain and be transmitted through the four or five upper dorsal ganglia. It is clear from this diagram why even complete removal of the cervical sympathetic ganglia may fail to relieve anginal pain. Even if all three cervical ganglia are removed, there still remain these nerves running to the five upper dorsal ganglia which may convey pain impulses. These can be completely interrupted by blocking the ganglia, the rami or the spinal roots.

These anatomical and physiological premises have been tested in a total of forty cases at the Massachusetts General Hospital suffering from the severest forms of anginal pain. Thirty-six of these had alcohol injections into the dorsal nerve roots or ganglia, and four were subjected to surgical resection of the upper four thoracic ganglia. Of the total number, there was complete or almost complete improvement (90 to 100 per cent relief) in more than half, and marked improvement (estimated at 50 to 90 per cent relief) in another 25 per cent. There was slight improvement (estimated at 25 to 50 per cent) in 8 per cent, and failure, due to technical difficulties in placing the alcohol precisely, in

about 12 per cent. There was no operative mortality from the paravertebral alcohol injections, but White estimates that there will be an inevitable mortality of about 25 per cent from surgical resection of the upper four thoracic ganglia.

Dr. Robert Levy, whose excellent paper upon this subject in 1931[4] led me to write him several weeks ago, has been kind enough to send me the following informal report upon the Presbyterian Hospital series of cases of alcohol injection for the relief of anginal pain. There have been about thirty cases in all; in approximately 40 per cent there has been a considerable measure of relief, varying from complete disappearance of pain to a reduction in the number and intensity of attacks. In another 30 per cent lesser degrees of improvement were noted, and in the remaining 30 per cent the procedure was classified as a failure. These figures are approximate, and represent Dr. Levy's impressions rather than a careful analysis of his cases. I would ask you to bear in mind that the differences in results at different clinics are not to be construed as invalidating the anatomical and physiological studies of White, but rather as indicating differences in experience and in technical skill. Several competent surgeons have told me, for example, that they are unable to perform the injection with any degree of success, and Dr. White tells me his technique is acceptable to himself only when he can perform injections on the average of about once a week.

Swetlow speaks of the relief in his patients as lasting for a number of months, but both White and Levy have found that there was no recurrence of pain in their patients who were relieved, and some of these have been followed for five or six years.

When we realize that the patients subjected to paravertebral alcohol injections have been selected purely on the basis of severity and frequency of pain, that they were critically ill, and that life was almost insupportable, these results are most impressive. There are, however, several objections that must be mentioned. The chief of these is the

appearance, in about one-half to two-thirds of the cases, of a severe alcoholic neuritis of the infiltrated intercostal nerves, leading to moderate or severe hyperesthesia and pain. This may last from several weeks to several months, and in a few cases is quite distressing. The second objection is the technical difficulty of injecting the alcohol with precision, since it has to be done blindly. The injection of 5 c.c. of alcohol results in an area of fibrosis about 1 cm. in diameter, and if this is to be effective, it must involve the exact area desired. In several reported instances, the alcohol has touched the pleura or has been injected into the pleural cavity and has led to transient pleurisy. In general, however, it must be regarded as a safe surgical procedure, without serious permanent complications, and without operative mortality.

There is one question which inevitably and quite properly comes to mind in this connection. Is it dangerous to abolish pain in these patients? Is anginal pain truly a warning, the removal of which will permit the patient to continue harmful exertion beyond the proper point and thus cause death? This question was vehemently debated in connection with the operation of cervical sympathectomy, but so far as I am aware, no clear evidence was ever brought forward in proof of the belief that patients would be worse without their warning pain. Similarly, I know of no evidence to indicate that alcohol injections have hastened death by permitting patients to indulge in excessive activity. Actually, the question is one that is very difficult, and perhaps impossible, to answer, for the patients who have been subjected to these procedures are precisely those to whom sudden death may come at any time. If they die suddenly some weeks or months after operation, who can assert with any confidence that the end has been hastened or postponed by the treatment? Actually, it is true to say that in most, if not in all, cases, there are substitution symptoms which serve as warnings just as effectively as did pain before its removal; such symptoms as a sense of emptiness in the epigastrium, oppression in the chest, or slight constriction of the throat.

I come now to the third and last of the surgical methods of treatment, namely, removal of the entire thyroid gland. I believe the largest experience with this method of treatment has been acquired by Levine and Cutler at the Peter Bent Brigham Hospital, and by Blumgart and his collaborators at the Beth Israel Hospital, in Boston. Most of you are doubtless familiar with their papers and with the slightly divergent views which they champion. Within the past two weeks I have had the privilege of discussing this matter at length with Doctors Blumgart, Davis and Riseman at the Beth Israel Hospital, with Doctors Cutler and Levine at the Peter Bent Brigham Hospital, with Doctors Weiss and Faulkner at the Boston City Hospital, and with Doctors Paul White, Howard Sprague and James White at the Massachusetts General Hospital. While my brief remarks upon the subject are based largely upon published reports, they are doubtless colored by my recent discussions with these men and by my own experience with the method in New Haven.

There is general agreement that during or immediately after such operation, the majority of patients are relieved of anginal pain. According to Blumgart, this instant relief is due to section of or trauma to sympathetic nerves in close proximity to the thyroid gland, and pain will return when the nerves regain their function. According to Levine and Cutler, the immediate relief is due to an altered response of the heart to adrenalin, and they have shown that the response of the cat's denervated heart to adrenalin is altered by removal of the thyroid gland from such animals. Blumgart, however, has shown that if only one lobe of the thyroid be removed from a patient who has angina with bilateral radiation of pain, there is immediate relief of pain on the operated side only. It is difficult to believe that this unilateral relief could be due to altered sensitivity to adrenalin circulating in the blood stream. Furthermore, Blumgart has shown that the response of the heart rate and blood pressure to intravenous injections of dilute adrenalin in man are precisely the same after thyroidectomy as before, until the basal metabolic rate falls to very low levels, which

usually requires three or four weeks. It is his conception that the immediate relief is due to cutting of nerves, and the permanent relief is associated with the lessened demands upon the heart attendant upon the development of hypothyroidism. His views seem so logical, and the evidence supporting them seems so unequivocal, that one is tempted to agree with them wholly, but there are many questions still unanswered, and further experience alone will provide the solutions that we seek.

What results have been obtained? I believe it is impossible at present to make any fair or final answer. Unquestionably some patients have been completely relieved of pain, and a larger number have been greatly helped. There have been a considerable number of failures, and a small number of operative deaths. Some patients have been relieved of pain, only to suffer the severe discomforts of myxedema, while others oscillate back and forth between myxedema and angina, depending on the dose of thyroid extract administred. So far as actual figures are concerned, Blumgart[14] reports that eight of twenty-five patients secured complete relief from pain lasting up to the time of publication of his paper, which was from three to eighteen months after operation. Five were improved, four had no relief, and six had recurrence of anginal pain after from three to nine months of complete relief. These figures in themselves seem to indicate that the physiological basis for the procedure is probably not fully understood. If relief of pain is actually due to lessened demands upon the heart associated with hypothyroidism, why should only eight of twenty-five patients secure lasting relief when hypothyroidism is produced? Why should six patients be completely relieved of pain, only to have it return six months later while the basal metabolism was still at a low level? Why do some patients secure relief from pain only when the metabolic rate falls to low levels, and have recurrences of pain if the metabolism is elevated even slightly, while others secure relief in a similar fashion and yet have no recurrences of pain even if the metabolism is raised to the preoperative level by means of thyroid extract? The evidence

in some respects is not only confusing, but also contradictory, and if the reported observations are correct, there can be little question that the evidence is still far from complete. It is possible that Cutler is right in believing that thyroidectomy has influences upon the circulation other than, and independent of, those which can be measured in terms of the basal metabolism. Until further studies can be completed, it seems impossible to state dogmatically the full reasons for the benefit which occurs in about one-third of the cases.

Moreover, there are certain surgical hazards which cannot possibly be ignored. The chief of these are sudden death within a few hours of operation, postoperative pulmonary complications which may lead to death several days later, injury to the recurrent laryngeal nerve, and parathyroid insufficiency. Unless these patients are treated with extreme care, the operative and postoperative mortality is apt to be rather high.

It was natural to believe, when this operation was first proposed, that serious results might follow the deliberate induction of surgical myxedema. Among these were mentioned the so-called myxedema heart, in the sense of a causal condition precipitating circulatory failure or angina pectoris, mental changes similar to those often associated with spontaneous myxedema, rapid development of arteriosclerosis because of the high level of cholesterol in the blood serum associated with hypothyroidism, and the anemia which is so often a part of spontaneous myxedema. These possible developments have been kept clearly in mind by Blumgart, and a large number of patients have been carefully studied to see if these conditions would develop. Up to the present time there is no evidence that they have been important. Despite this, the potential and actual disadvantages of thyroidectomy are numerous, and there is complete agreement among all those with whom I have talked that a certain number of patients have been made worse by the operation, not with respect to pain, but with respect to other symptoms. The discomforts and crippling effects of myxedema are too numerous and serious to be ignored, and

the final state of some patients after operation is most pitiable. It seems pertinent to indicate that with the exception of the Beth Israel group, those with whom I have discussed the matter are far less enthusiastic today than they were a year ago.

These I believe to be the chief matters for consideration in attempting to appraise the value of these surgical procedures individually and with respect to each other. I have already indicated that there is now a general belief that operations upon the cervical sympathetic trunk are physiologically unsound and not warranted in the light of present knowledge.

A comparison of the other two methods may be attempted, but any conclusions now reached may well be modified by future experience. It should be kept clearly in mind that the two have quite different aims; the injection of alcohol has as its objective the destruction of nerves which convey pain; in a sense it aims at producing permanent local anesthesia. Thyroidectomy, on the other hand, theoretically aims at removing the discrepancy between the demands of the body and the ability of the heart to meet those demands. Anginal pain is thought to be the expression of this discrepancy, and since the ability of the heart cannot be increased in terms of the coronary blood flow, it would seem logical to decrease the demands of the body, and thus remove the cause of the pain. There is no apparent reason for questioning the physiological basis for the first procedure; there are several reasons for questioning the current explanations of the relief afforded by thyroidectomy. Until we know beyond all question exactly what thyroidectomy does, the only comparison that is valid is of the results achieved by the two methods, both good and bad, and of the obvious advantages and disadvantages of each. Accepting the reported results as published or as reported verbally to me, the percentage of relief following alcohol injections is higher than that following thyroidectomy, at least in the Boston groups. Further advantages of this procedure are the absence of operative mortality, the absence of any per-

manent ill-effects, the short period of hospitalization (usually three or four days, as compared with several weeks for thyroidectomy), and the possibility that other methods may be tried in case of failure, without the patient's condition having been altered for the worse. The disadvantages have already been indicated; they are chiefly three: the technical difficulty of injecting the alcohol accurately, the neuritis that often follows it, and the fact that relief is strictly limited to the side on which injection is performed. . . . In favor of thyroidectomy one may say that it affords great or moderate relief in one-third to one-half of the patients, including those who have bilateral radiation of pain. So far as I know, this is its only actual advantage as compared with the other procedure. The disadvantages are numerous and have been mentioned briefly; they include operative and postoperative death, recurrent laryngeal nerve paralysis, parathyroid insufficiency, myxedema, and the disturbing fact that we are still uncertain of the physiological basis for the operation.

A final question may profitably be asked. Is there a place for either or both of these therapeutic procedures in the treatment of anginal heart failure? In my opinion there is most certainly a place for alcohol injections. I believe the present evidence indicates that thyroidectomy will also find a permanent place in the treatment of angina, although I share the prevailing belief that at present it should be reserved for those patients in whom pain makes life intolerable or prevents the earning of a livelihood. The criteria for the selection of suitable patients are not yet sufficiently known, and it is possible that it will ultimately be reserved for a few severely ill patients who have bilateral radiation of pain, who have hyperthyroidism in addition to angina, or who have failed to respond to alcohol injections. The injection of alcohol would seem to me the method of first choice because of the high incidence of success and its freedom from the distressing conditions which may follow removal of the thyroid gland.

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DISCUSSIONS

DR. ALEXANDER LAMBERT: That was an extremely interesting and instructive summary that Dr. Marvin just finished; and one which I followed with great pleasure. It was on a subject in which I had long been interested, and I had followed closely the literature as it had appeared. As he summed up the thyroid operation for angina pectoris, it reminded me of the old Greek aphorism that was said of opium, "opium cures a disease, and produces a disease." I think this surgical procedure does the same. The elder Kocher, years ago, found that total ablation of the thyroid left a condition in which the several forms of thyroid medication could not prevent myxedema, and with it the myxedematous heart.

I do not see that it fulfills the entire requirements for the relief of angina to remove a gland because its secretion is a stimulant of the sympathetic system, but leaves the sympathetic system itself intact. The pains of angina are brought about by disturbance of the normal equilibrium between the two balancing parts of the autonomic nervous system, and the three chief conditions which bring on attacks of angina are emotion, exertion and exposure to cold. These three conditions are sympathetic drives to redistribute the blood in the body. The heart normally is under the control of the vagal system, and the blood vessels under the sympathetic. When a sympathetic drive occurs requiring redistribution of blood, a heart in good condition increases its output per beat, but a heart out of condition cannot do this immediately, or even at all, and must endeavor to meet the demand for more blood by an increase of rate to give an increase per minute. Pain occurs when the sympathetic system demands a greater increase of cardiac function than the heart can supply. Cutting out the thyroid gland to diminish one of the chief stimulants of the sympathetic mechanism leaving an intact sympathetic system is only a partial answer to the problem of cardiac pain production.

There is one method of bringing the autonomic system back into equilibrium that has recently been brought more and more into prominence, and which has been proven effective in Europe for many years, but which in this country is held in very little repute. That is the carbonic acid baths as given at Saratoga Springs, New York, at Nauheim, St. Moritz, and other places abroad. These baths, as has been recently shown by Bornstein, Budelmann and Rönnell, do return the control of the heart to the vagal system, as shown by diminution of pulse rate, and by increasing the power of the heart to deliver an increase of blood through each beat of the heart. It has also been shown by Voigt that hot plain water baths increase the cardiac output, but chiefly by accelerating the heart through the sympathetic system, and increasing the output by increasing the output per minute. This is the basic result of those baths.

I was asked to speak upon the effect of tobacco in angina, and the clinical effect of tobacco in the ordinary life of the patient with angina. Clinically, patients having angina pectoris, and who use tobacco are more prone to attacks, and more prone to increase the intensity of their pain during the attack, than those with angina who do not use tobacco. That is a curious fact, but it is so. Some patients can smoke with impunity, with no injurious effect, even though they have angina. Other patients have to give up smoking. I have seen patients who have given it up and returned to it who dropped dead after their first cigar. I have seen only five of such accidents, but this has been enough to have attracted my attention.

Harkavy and Sulzberger reporting their tobacco experiments find in the thrombo-angiitis patients, that over 80 per cent showed a marked sensitization to tobacco in the skin. In the 319 control smokers who were without the thrombo-angiitis there were only about 20 per cent who showed this tobacco reaction. About one-third of these also showed a family history of asthma, and hay fever. It is also interesting to note that practically one-half of the smokers who showed skin reaction to tobacco, also showed the symptoms of angina pectoris. In the other controls there were only about 12 per cent who showed a skin reaction to tobacco. This reaction is not due to nicotine. The skin reaction due to nicotine appears in but a very small number of patients. The reaction is due to some extract from the tobacco itself which has not been separated. It may show in some patients, in some certain forms of tobacco only, or in some one brand of cigarettes, but not in others, and be entirely peculiar to that person. It is here a matter between a patient and his brand of tobacco. Other patients showed skin reaction to all forms of tobacco tested.

It has long been noticed that nicotine paralyses the synapses of the sympathetic nerves between the pre-ganglionic and post-ganglionic fibers. As you know, the action of the autonomic nervous system is produced by the secretion from the nerve endings themselves of certain chemical sub-

stances, the parasympathetic nerves secreting acetylcholin, and the sympathetic nerves, a substance called by Cannon, sympathin.

Nicotine and atropine prevent the action of these secretions in these nerves. Atropine, for instance, does not act directly on the nerve endings of the parasympathetic, and nicotine does not act directly on the nerve endings, but they act chemically on the acetylcholin, or sympathin as secreted, and prevent their action. Nicotine thus inhibits the action of the sympathetic nerves as cardiac accelerators, and also diminishes the conductivity and contractility of the myocardial fibers in the cardiac ventricle.

Thus when nicotine does affect the sympathetic action of the heart, it has a direct action on the nerves governing the contractility of the ventricular muscles.

Regnier has shown, studying Tschermak's test by pressing the carotid sinus in the neck, that in patients suffering from arteriosclerosis, or those having myocardial degeneration, the heart is excessively sensitive to disturbance of the nervous reactions produced by this pressure test.

It seems that in the senile heart, as we may call it, there is an increased sensitivity to its nervous mechanism, and it may well be that the disturbances of the mechanism causing the sensation of pain in the heart, are more often due to the use of tobacco than is usually acknowledged.

It is wise, therefore, to take seriously the possibilities following indications of the disturbance in the synapses between the post-ganglion and pre-ganglion fibers, by nicotine.

DR. MARCUS A. ROTHSCHILD: I have been asked to confine my discussion to the question of the mechanism of angina, especially the anoxemic hypothesis. In all probability too much emphasis has been laid on the purely mechanistic idea of the production of pain in angina pectoris. We probably have to go back to something more

fundamental and think, possibly, in terms of the humoral theory of the transmission of nerve impulse.

I do not personally believe that the pain in angina pectoris is due entirely to a disproportion between the amount of work asked of the heart to perform and the amount of blood the muscle receives. There is a nerve element in the whole process. There are many reflexes which will induce the reflex, as I should like to call it, angina pectoris. For example, simply cooling a single arm of a susceptible individual, or by lowering the temperature of the room to a certain degree. In other people, a distended stomach, 2 or 3 glasses of water; in others, performing a limited quantity of work will induce the attack. Some individuals may walk up three flights of stairs in their own home without an attack, but will be unable to walk two blocks in the street. It is actually not the number of foot pounds of work performed that induces the attack:—there are other elements involved.

We were interested in attempting to produce the attacks by means of induced anoxemia. We felt that if the segments of the coronary system were narrowed that by inducing a general anoxemia we might induce a local anoxemia in that area of heart muscle supplied by the narrowed vessel. It was thus assumed that this area of heart muscle would receive an inadequate supply of oxygen and nutrition. We induced the anoxemia by having the patient rebreathe with a sufficiently large rebreathing apparatus and CO_2 absorbing chamber so that the cerebral effects would be reduced to a minimum and would not antedate the general anoxemic effects. Briefly, we succeeded in inducing pain in 18 out of 24 subjects diagnosed as having coronary disease. The attacks were identical in nature with those the patient spontaneously complained of; i.e., if the patient spontaneously began with pain in the elbow, the induced anoxemic attack began with pain in the elbow. We have performed this test on several hundred occasions and can report no harmful results. In two instances the attack persisted after the patient was removed from the tent and it required a vasodilator in order to remove the attack.

Associated with the production of these attacks by means of anoxemia we found in a small percentage of cases changes in the electrocardiogram, but in a certain number of normal individuals—normal as far as we could determine—similar changes were found.

It is an interesting fact, however, that after the patient has had an attack of coronary artery thrombosis, many of the cases lose their pain. We have tried to interpret this on the basis that the diseased vessel being completely occluded, the area of heart muscle supply to this vessel being thrown out of circuit, no pain reflex could arise from that area. The reappearance of pain would seem to indicate that another branch of the coronary system was being involved. We are at present investigating this phase of the subject.

Dr. Kissin continued this work on the skeletal muscle and found the same general principles to be true. In the normal individual performing a certain amount of work with his fingers, subjecting him to anoxemia, he could do less work and pain would follow if the work were persisted in. It seems interesting that the skeletal muscle in normal individuals gives a pain reflex while it is impossible to produce the pain in normal heart muscle by means of any grade of anoxemia which we have been able to produce, and we have been close to six volumes percentage. The lines of investigation in this work may throw some light on the mechanism of a certain number of cases.

I do not believe that anoxemia is the single explanation of all cases of angina. I think it is necessary to go very deeply into the metabolism of the muscle and attempt to learn how anoxemia for example brings about the reflex. It is not purely the lack of oxygen. There is some chemical process involved similar to the type of neuro-chemical process involved with the induction of attacks of pain by the cooling of an arm or by an emotional state.

DR. JOHN WYCKOFF: Mr. President, Ladies and Gentlemen: I think we have been very fortunate to hear these three papers. I cannot discuss Dr. Marvin's paper, but I would like to say, as did Dr. Pool, that I never heard a more masterly, thorough or well-weighted discussion of a controversial subject. The Academy, I think, has honored itself tonight in naming Dr. Libman a benefactor of the Academy. I wonder if the profession realizes how much a benefactor he has been for a great many years to the profession at large? The subject on which he spoke so very ably tonight is one of his great benefactions. It was, of course, due to the work of Dr. Libman and the work of Dr. Herrick of Chicago that coronary thrombosis came to be identified in our minds with angina pectoris in a large number of cases. There is not any one who could present the subject to us in such a masterly way as he has.

There are two or three things which he mentioned that I would like very briefly to touch upon. He spoke of the fact of multiple thrombi and the tendency of individuals to have such multiple thrombi. This subject has interested some of us working at Bellevue. Dr. Irving Graef and Dr. de La Chapelle have been particularly interested in this phase of the subject. It has been rather a difficult thing to study. They have felt that hearts in which a thrombosis had occurred in the ventricle should be eliminated because of the fact that an embolus might break off from such a thrombus and thrombi found elsewhere might not be spontaneous thrombi, but due to embolism.

During the last year or two, ten hearts have been studied in which a coronary thrombosis had occurred without any ventricular thrombus. Nine of these ten hearts show that there is a thrombus elsewhere in the vascular system. Three show more than one coronary to be involved. In two of the cases there was thrombosis of the aorta; in one a thrombus in the right auricle; in two there was, besides coronary thrombosis, thrombosis of the pulmonary artery; in one there was renal and splenic thrombosis besides coronary thrombosis.

All of this is very interesting and bears out Dr. Libman's observations. He also spoke of the fact that not all patients having coronary thrombosis have pain. In the last 23 cases studied by us that have died after their attack and have been autopsied, only nine had severe and typical anginal pain. Six of them had epigastric distress, but this was not the typical anginal pain so frequently associated in our minds with coronary thrombosis. Four of these patients had no other symptoms than the symptoms of shock. All 23 of the patients, however, (none of whom died immediately during the attack) did have dyspnœa.

I think we are always very fortunate to hear from Dr. Brooks, particularly when he talks on the treatment of any illness. He always keeps his feet so firmly on the ground.

There are two or three things that I think those of us who take care of patients with heart pain should bear in mind, and one, which Dr. Trudell spoke of as being so necessary to patients having tuberculosis, is a constant spirit of optimism. These patients not only have a serious disease which very frequently is on an organic basis, but they have a disease, the symptoms of which are greatly accentuated by mental trauma and nothing can do more harm to the patient than to handle his psychology improperly. It seems to me that too many doctors are unwilling to take the responsibility, and I think it is the doctor's responsibility, of being optimistic and letting people feel that things are not quite so bad as they perhaps are.

I also think that this is a disease in which a doctor must have a certain sureness of touch. He must not fumble. I think these simple things Dr. Brooks has brought out very well. I would also like to put in my little word for alcohol, in spite of the fact that Dr. Lambert is on the platform, and say that I know of hardly any drug more useful in the handling of patients who have this symptom than the proper use of alcohol.

I might say just one word about the difficulty which sometimes arises in making the diagnosis of coronary thrombosis, because of the onset of rapid heart action.

When a coronary is thrombosed and there is a sudden development of anoxemia, not infrequently there develops a paroxysmal tachycardia which is frequently taken for, and frequently is, a symptom of coronary thrombosis. The reverse is true. Sometimes a patient with a heart which is nearly normal will develop an attack of rapid heart action, for example, an attack of paroxysmal tachycardia, and with the attack will develop a great deal of precordial pain. It does not have to be a great deal of precordial pain to be very confusing. Of course, one is a very serious condition and the other is not very serious. I think we should always bear in mind when we see a patient with a sudden attack of rapid heart action that he need not necessarily be having an attack of coronary thrombosis. I think of late our minds have been moving along that line and sometimes such a diagnosis is made a little too frequently.

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HUTCHINSON, ROBERT HAMILTON, Jr., M.D., 166 West 87 Street, New York City; graduated in medicine from the College of Physicians and Surgeons in 1905; elected a Fellow of the Academy March 1, 1928; died June 21, 1935. Dr. Hutchinson was a member of the County and State Medical Societies, and a Fellow of the American Medical Association and the American College of Surgeons. He was Assistant Professor of Clinical Otolaryngology in the Post-Graduate Medical School and Junior Surgeon to Bronx Eye and Ear Infirmary.

TYSON, CORNELIUS JOHN, M.D., 37 East 64 Street, New York City; graduated in medicine from Bellevue Hospital Medical College in 1912; elected a Fellow of the Academy November 6, 1919; died June 28, 1935. Dr. Tyson was a member of the County and State Medical Societies and a Fellow of the American Medical Association. At the time of his death Dr. Tyson was president of the medical board of St. Vincent's Hospital and director of its medical division. He was consulting physician to French and Southside Hospitals.

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